AD-A283 092

PAGE

Form Approved
OMB No. 0704-0188

1. AGENCY USE ONLY (Leave blank)	2. REPORT DATE	3. REPORT TYPE AND DATES COVERED
	July 1994	Final

4. TITLE AND SUBTITLE
Arthropods: Vectors of Disease Agents

5. FUNDING NUMBERS
PR - 6750
TA - 32
WU - IX

6. AUTHOR(S)
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7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)
Occupational and Environmental Health Directorate,
Armstrong Laboratory
Brooks AFB TX 78235-5114

8. PERFORMING ORGANIZATION REPORT NUMBER
AL/OE-JA-1993-0108

9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)

10. SPONSORING/MONITORING AGENCY REPORT NUMBER

11. SUPPLEMENTARY NOTES
LABORATORY MEDICINE Vol.25, No. 7, July 1994

12a. DISTRIBUTION/AVAILABILITY STATEMENT
Approved for public release; distribution is unlimited.

12b. DISTRIBUTION CODE

13. ABSTRACT *(Maximum 200 words)*

This is the third and final article in a continuing education series on arthropods. The interactions between arthropods and the various etiologic agents they transmit are reviewed. The incidence of arthropod-borne diseases in the United States, the causative agents, and their reservoirs, vectors and transmission mechanisms are summarized in a Table. The ecology of five arthropod-borne diseases - Lyme disease, eastern equine encephalitis, plague, tularemia, and leishmaniasis - is discussed to illustrate the complex, dynamic nature of these disease cycles. The evidence for the lack of arthropod transmission of HIV is examined. The clinical importance of arthropod identification and limited prognostic value of screening for etiologic agents are covered.

14. SUBJECT TERMS
arthropod-borne disease, vector, ecology, epidemiology

15. NUMBER OF PAGES
9

16. PRICE CODE

17. SECURITY CLASSIFICATION OF REPORT	18. SECURITY CLASSIFICATION OF THIS PAGE	19. SECURITY CLASSIFICATION OF ABSTRACT	20. LIMITATION OF ABSTRACT
Unclassified	Unclassified	Unclassified	UL

Arthropods: Vectors of Disease Agents

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Reprinted from LABORATORY MEDICINE
Vol. 25, No. 7, July 1994.

Arthropods: Vectors of Disease Agents

Chad P. McHugh, MPH, PhD

This is the third and final article in a continuing education update series about arthropods. The first article explored identification of medically important arthropods. The second article dealt with how arthropods directly affect human health. This article highlights the role of arthropods as transmitters of disease agents. In mechanical transmission, pathogens may be transmitted to the host through the vector's mouthparts or feet or be passively regurgitated during blood-feeding. Biological transmission requires that pathogens go through a cycle of development or multiplication in the vector's body before being transmitted to the host. In the United States, zoonoses are the most commonly diagnosed arthropod-borne diseases, with tick-borne diseases particularly common.

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Arthropod-borne diseases (ABDs) or, more correctly, etiologic agents transmitted by arthropods cause significant sickness and death worldwide. Each year, 1 to 2 million people die of malaria. This is just a small percentage of the 107 million new cases diagnosed per year. At least 12 million people throughout the world are infected with *Leishmania* species, which exist on every continent except Australia and Antarctica. An estimated 50 million and 90 million people are at risk of African trypanosomiasis (sleeping sickness) and American trypanosomiasis (Chagas' disease), respectively.[1]

Although ABDs are not as prevalent in the United States as they are in many tropical countries, cases do occur here (Table). Today, the most commonly diagnosed ABDs in the United States are zoonoses, animal diseases that are occasionally transmitted to humans. Tick-borne zoonoses are particularly important. The clinical presentation, diagnosis, and treatment of several tick-borne diseases have been reviewed recently.[2] The incidence of some historically important diseases such as malaria has declined, in part because of improved housing, health care, and arthropod control programs, but imported and secondary, introduced cases of this and other exotic diseases continue to occur.

Arthropod-Pathogen Interactions

In each of these disease cycles, an arthropod serves as the vector transferring the etiologic agent from an infected reservoir to other hosts. Relationships between pathogens and their vectors are varied and often

Test Time

See page 465 of this issue for the continuing education update exam on arthropods.

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Arthropod-Borne Diseases of Historical or Current Importance in the United States*

Disease	Pathogen(s)	Vector(s)
Tick or mite-borne		
Powassan encephalitis	Flavivirus	Ixodid ticks
Colorado tick fever	Orbivirus	*Dermacentor andersoni*
Q-fever	*Coxiella burnetti*	*Dermacentor andersoni, Rhipicephalus sanguineus*
Rocky Mountain spotted fever	*Rickettsia rickettsii*	*Dermacentor andersoni, D variabilis*
Rickettsial pox (mite-borne)	*Rickettsia akari*	*Liponyssoides sanguineus*
Relapsing fever	*Borrelia turicatae B hermsii, B parkeri*	*Ornithodoros* spp
Lyme borreliosis	*Borrelia burgdorferi*	*Ixodes scapularis, Ixodes pacificus, Ixodes* spp
Ehrlichiosis	*Ehrlichia chaffeensis*, other *Ehrlichia* spp?	Ixodid ticks
Babesiosis	*Babesia microti*	*Ixodes scapularis*
Mosquito-borne		
St. Louis encephalitis	Flavivirus	*Culex* spp
Western equine encephalitis	Alphavirus	*Culex* spp
Eastern equine encephalitis	Alphavirus	*Culiseta melanura, Aedes* spp, *Aedes albopictus?*
Venezuelan equine encephalitis	Alphavirus	*Aedes, Psorophora*
La Crosse encephalitis	Bunyavirus	*Aedes triseriatus*
Dengue	Flavivirus	*Aedes aegypti, Aedes albopictus?*
Yellow fever	Flavivirus	*Aedes aegypti, Aedes albopictus?*
Malaria	*Plasmodium* spp	*Anopheles* spp
Dirofilariasis	*Dirofilaria immitis*	*Aedes, Anopheles, Culex*
Fly-borne (other than mosquito)		
Leishmaniasis	*Leishmania mexicana*	*Lutzomyia anthophora, Lutzomyia diabolica*
Flea-borne		
Plague	*Yersinia pestis*	Fleas (many species)
Murine typhus	*Rickettsia typhi*	*Xenopsylla cheopis*, others
ELB agent	*R typhi*-like	*Ctenocephalides felis*
Louse-borne		
Epidemic typhus	*Rickettsia prowazekii*	*Pediculus humanus*
Relapsing fever	*Borrelia recurrentis*	*Pediculus humanus*
Other		
Tularemia	*Francisella tularensis*	Ixodid ticks, biting flies
Chagas' disease	*Trypanosoma cruzi*	Kissing bugs (reduviids)
Enteric diseases	*Salmonella, Shigella*, others	Roaches, filth flies, ants, others?

* Question marks indicate possible association.
† Incident cases, 1983–1992, data for other periods shown parenthetically. NA, data not available, not reportable, uncommon diseases, or a combination.
‡ Alternate modes of transmission, etc.
§ California group encephalitis, most of which were due to LaCrosse encephalitis virus.
¶ Includes cases in Puerto Rico.

Reservoir(s)	Transmission Mechanism(s)	Human cases† 1983–1992 (or other)	Remarks‡
Rodents, carnivores, rabbits	Biological (transstadial)	NA	Rare, raw goat's milk
Rodents	Biological (transstadial)	(200–300/yr)	Biphasic fever
Domestic livestock ticks	Biological (transstadial, transovarial)	NA	Airborne, milk, direct contact
Dermacentor spp.	Biological (transstadial, transovarial)	(600–1,000/yr)	Maculopapular rash on palm & soles, spreads to trunk
House mouse	Biological	NA	Few cases in New York City
Rodents, ticks	Biological (transstadial, transovarial)	(113, 1985–1989)	Ticks long-lived, infective for years
Deer mice, wood rats	Biological (transstadial, transovarial)	49,375	Primarily northeast and midwest US
Rodents?	Biological	320	Bullis fever?
Deer mice	Biological	NA	Flulike illness
Birds	Biological	512	Occasionally large outbreaks
Birds	Biological	60	
Birds	Biological	54	Isolated cases, "mini-epidemics"
Rodents	Biological	None in past decade	
Rodents, rabbits, *Ae triseriatus*	Biological (transovarial)	610§	
Humans	Biological	(1,095, 1982–1991¶)	4 serotypes, "breakbone fever," epidemics of 1000s
Humans, monkeys	Biological	NA	
Humans	Biological	9,957 imported, 20 congenital, 21 induced, 75 introduced or cryptic	4 species
Canines	Biological	NA	Coin lesions in lung mimic carcinoma
Neotoma micropus (possum, cotton rat, armadillo?)	Biological	<30	*L diabolica* as vector to humans
Rodents (many species)	Mechanical, biological	154	Enzootic-mechanical, Epizootic-biological
Rats, mice	Biological	NA	Milder than epidemic typhus
Ctenocephalides felis	Biological (transovarial)	NA	Newly identified agent, human pathogen
Humans, flying squirrels?	Biological	NA	Epidemic during wars, recrudescence = Brill-Zinsser disease
Humans	Biological	NA	
Rabbits, rodents	Biological (ticks), mechanical (flies)	2,006	Direct contact, food-, dust-, & water-borne
Wood rats, opossums, racoons, armadillos	Biological	<5	More common in dogs than people
Garbage, sewage, etc	Mechanical	NA	

A sand fly feeding on a human host. Wings are held in a "V" shape over the body (courtesy Dr E. Rowton).

complex. These relationships may be mechanical or biological.

Mechanical Transmission

In mechanical transmission, the pathogen may contaminate the mouthparts or feet of the vector or be passively regurgitated during blood-feeding. For example, flies can transmit tularemia and the enteric pathogens. Transmission requires survival of the pathogen, but not its biological development or multiplication in the vector. The vector serves merely as a "living fomite" facilitating the between-host transfer.

Biological Transmission

In biological transmission, the pathogens must undergo a cycle of development (cyclodevelopmental transmission), multiplication (propagative transmission), or both (cyclopropagative transmission) before transmission can occur. Enormous variation exists in the location within the vector where the cycle occurs and the route by which pathogens exit the vector.

Most arthropod-borne viruses (arboviruses) penetrate the gut wall of the vector, multiply in body tissue, invade the salivary glands, and are injected into new hosts during feeding. *Borrelia* spp (tick-borne relapsing fever) multiply in the body tissues and are transmitted in the saliva of their tick vectors, but also are excreted onto the skin of the host in fluid from the coxal glands, which are special excretory glands in the tick legs. Filarid worms (eg, dog heartworm) develop in the vector tissues, but migrate to the mouthparts, actively break through the cuticle, and are deposited on the skin of the host in a drop of arthropod blood (hemolymph). The transmission cycles of the plague bacillus and *Leishmania* are limited to the gut of the vector. These pathogens block the gut and are regurgitated into the feeding wound, damage the feeding apparatus of the vector and leak out its mouth during feeding, or both.

The cycles of *Trypanosoma cruzi* (Chagas' disease), *Rickettsia prowazekii* (louse-borne typhus), and *Rickettsia typhi* (flea-borne typhus) also are limited to the gut (including the gut epithelium in the case of typhus), but they are passed in the feces and contaminate the skin, feeding wound, or mucosa of a host. *Borrelia recurrentis* (louse-borne relapsing fever) penetrates the gut wall and multiplies in the hemolymph, but not the tissue, of the louse. This pathogen is not found in the saliva or feces. Transmission occurs only when the louse is crushed and infected hemolymph contaminates the host's skin.

Arthropod-to-Arthropod Transmission

Transstadial. Some pathogens acquired in one life stage of an arthropod vector survive a molt to the arthropod's next life stage. This stage-to-stage (transstadial) transmission between arthropod life stages is important in vectors such as most hard tick (ixodid) species, which feed only once in each life stage. An infection acquired in one stage may be transmitted to other vertebrate hosts in subsequent stages.

Parent-to-Offspring. Pathogens that penetrate the arthropod gut and multiply in arthropod tissues may invade the ovaries, be included in the eggs, and result in infected offspring.

This parent-to-offspring (transovarial) transmission is important in some tick species, individuals of which feed on only one host during their entire life. In these species, parental ticks acquire an infection that is transmitted to new hosts by the following generation.

In mosquitoes, transovarial transmission obviates the need for an initial, infective bloodmeal or an incubation period in the vector, thus increasing the number of times a vector may transmit pathogens.

If transovarial transmission is very efficient (ie, a high percentage of offspring are infected), it may allow for the long-term survival of the pathogen, even in the absence of infected vertebrate hosts. For these pathogens (eg, La Crosse encephalitis virus and Rocky Mountain spotted fever), the arthropod may serve as a reservoir as well as vector of the agent.

The Ideal Vector

In addition to providing a suitable environment for the pathogen (in the case of biological transmission), the ideal vector would be long lived, have a host

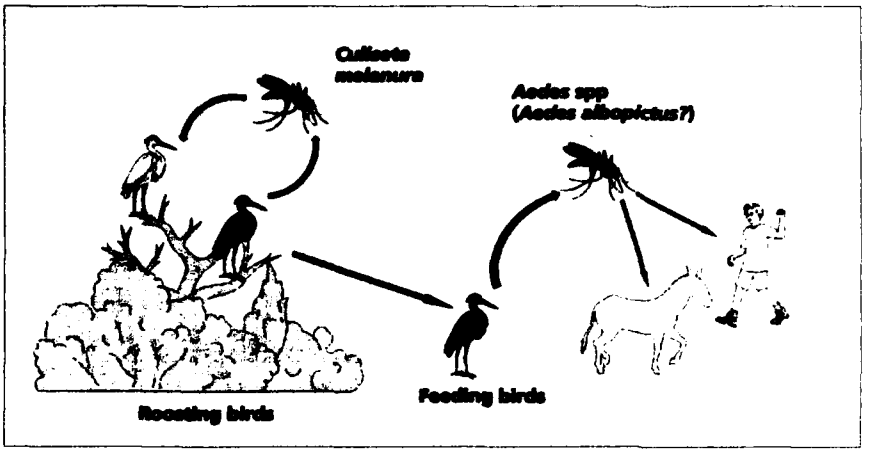

Fig 1.—Transmission of eastern equine encephalitis virus from infected hosts and vectors (solid figures) to susceptible hosts (line figures). Question mark indicates possible association.

feeding pattern that matches the host range of the pathogen, feed often and for extended periods, ingest large amounts of blood in each life stage, and disperse readily. No arthropod possesses all these characteristics, but for each disease in each geographic area, at least one "primary" vector possesses the characteristics necessary to maintain the transmission cycle. Vectors that transmit to a lesser extent or only under certain circumstances are termed "secondary" vectors.

Ecology of Arthropod-Borne Diseases

Arthropod-borne diseases, especially zoonotic ABDs, are complex, multifactorial diseases. Many ABDs have multiple hosts and vectors as well as alternate modes of transmission. To understand the ecology of ABD, one must understand not only the life history, behavior, and biology of the vectors, hosts, and pathogens, but also all interactions among them and environmental influences. In many cases, the ecology of these cycles is not completely understood, and, for the layperson, changes in scientific nomenclature of the vectors and pathogens may also make the cycles harder to understand. These are dynamic systems, and changes to any aspect of the disease cycle or the environment may lead to the emergence of "new" diseases, such as Lyme disease, or the resurgence of "old" diseases, such as malaria.

In the United States, the importance of ABD could increase dramatically if humans intrude environments with preexisting disease cycles, if exotic arthropods are introduced and serve as more efficient vectors of endemic pathogens, if land-use patterns or ecological changes allow endemic cycles to spread into other ecological settings, or if exotic pathogens are introduced into areas with susceptible hosts and vectors.[3,4]

The following transmission cycles represent emerging diseases, illustrate the complex nature of ABDs, or are examples of the various transmission patterns.

Eastern Equine Encephalitis

Of the four mosquito-borne encephalitides that occur in the United States—St Louis, eastern equine, western equine, and La Crosse—eastern equine encephalitis (EEE) is the most serious. About 50% of human cases result in death; neurologic sequelae result in a high percentage of survivors.

We do not yet completely understand the ecology of EEE virus. Transmission to humans appears to include two different cycles and several species of adult, female mosquitoes (Fig 1). The enzootic maintenance cycle (endemic in animals) involves transmission to roosting and nesting birds by *Culiseta melanura*. These mosquitoes breed only in densely vegetated swamps. Because of the mosquito's habitat limitation and restricted host preference, its role is limited to bird-to-bird transmission. When the birds leave roosting sites to feed in more open areas, they encounter other mosquitoes, such as *Aedes sollicitans, Aedes vexans, or Aedes taeniorhynchus*, which have a wider host range and may transmit EEE virus to humans during subsequent blood-feedings. Humans and horses are usually dead-end hosts for the virus.

Eastern equine encephalitis is uncommon in humans. Usually a "mini-epidemic" of only one or a few cases occurs. With the introduction of *Aedes albopictus*, an exotic mosquito believed to be imported from Asia in used-tire casings, this pattern may change. This mosquito is an aggressive human biter, a competent vector of EEE virus, and has spread rapidly over the eastern two fifths of the United States. *A albopictus* has become a serious pest in some areas of the southeastern United States and could be a very effective bridge between infected birds and susceptible humans. In 1991, EEE virus was isolated from *A albopictus* collected in central Florida.[5] If this species becomes routinely involved in the EEE virus cycle, we may see the number of human EEE cases increase dramatically.

Lyme Disease

Lyme disease, caused by the spirochete *Borrelia burgdorferi*, is a chronic, multisystem disease. The first symptom is typically a bull's-eye rash (erythema migrans), with the disease progressing to myocarditis, arthritis, and nervous system involvement.[6]

Lyme is a classic example of an emerging disease. First detailed in 1977 in patients from Old Lyme, Conn,[7] Lyme disease has become the predominant arthropod-borne disease in the United States.

Conditions leading to the emergence of Lyme borreliosis include land-use patterns that encourage

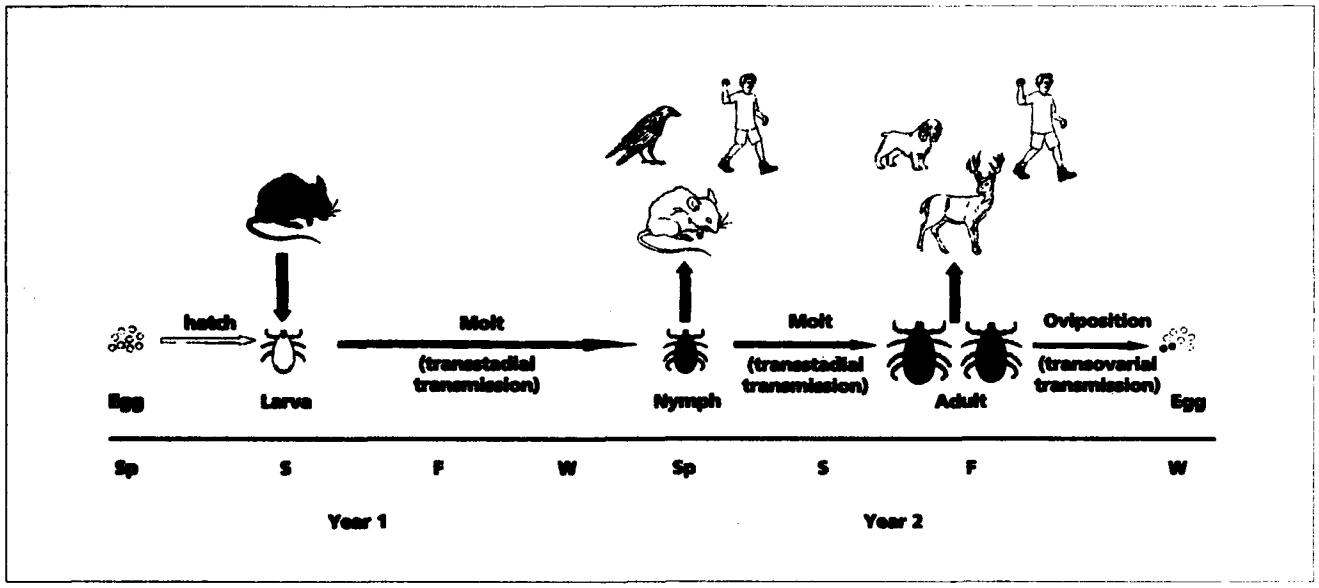

Fig 2.—Transmission of *Borrelia burgdorferi* from infected host and vectors (solid figures) to susceptible hosts (line figures) through the seasons for a 2-year period.

increased white-tail deer and deer tick populations, the increase in humans' outdoor activity, and intrusion of suburbs into deer habitat.

Although human cases occur in almost all states in the contiguous United States, the disease is most prevalent in the northeastern and midwestern states. *Ixodes scapularis* (*Ixodes dammini* is now considered conspecific with *I scapularis*) is the vector of *B burgdorferi* in this region. *Ixodes scapularis* has four life stages: egg, larva, nymph, and adult (Fig 2). Uninfected eggs hatch to uninfected, six-legged larvae that feed once to repletion in the late spring and summer. Small mammals, including the white-footed mouse, which serves as the reservoir of *B burgdorferi*, are preferred hosts.

Infected larvae molt and pass the infection (transstadial transmission) to eight-legged nymphal ticks. Nymphs survive the winter and feed once the following spring, infecting a new generation of mice that will serve as a source of *Borrelia* for larvae feeding later that summer. Most human infections probably result from feeding nymphs, which are small and difficult to detect. Nymphs molt and pass the infection to adult male and female ticks, which feed once, mate, and oviposit in the fall or early spring. Deer or other large mammals are preferred hosts; adult ticks feed less often on humans and are more easily seen and removed. The infection in both nymphal and adult ticks is limited to the midgut initially, but then disseminates to other tissues, including salivary glands, about 2 to 3 days after attachment. If the ovarioles of females become infected, *Borrelia* will be passed to a small proportion of their eggs (transovarial transmission).

The deer mouse–tick cycle maintains *Borrelia*, but tick abundance is related to deer availability. Strategies to control Lyme disease have thus evolved along two lines. Acaricides, applied directly to tick habitat or to material that mice will gather and use in their nests, reduce tick abundance directly. Exclusion or elimination of deer will reduce tick abundance, but the reduction requires several seasons and may initially increase tick-human contact as the preferred host is eliminated.

In the southern and western United States, the ecology of Lyme is not well understood, but differences in the tick and mammal

The sand fly *Lutzomyia anthophora* feeding on a woodrat host (courtesy Dr R.G. Endris). (From *Ann Entomol Soc.* 1981;74:[Cover]. ©1981 by the Entomological Society of America. Used by permission.)

species involved in the cycle may limit human exposure. In California, for example, *Ixodes pacificus* is a vector. Larvae of this tick species frequently feed on lizards, incompetent hosts for *Borrelia*, which reduces the number of infected nymphal ticks. A wood rat-tick cycle also may exist,[8] but the *Ixodes* in this cycle are closely associated with the wood rat nest and unlikely to encounter humans.

Plague

Yersenia pestis, the bacterial agent that causes plague, was introduced into California about 1900 and has since spread throughout the western United States. The plague bacillus has a number of hosts and transmission mechanisms (Fig 3).

The bacillus is transmitted among rodents and fleas, which are relatively resistant. The fleas do not develop an overwhelming infection and transmission is primarily mechanical.

When plague spreads into susceptible rodent populations, epizootics (epidemics among animals) with high mortality rates occur. Fleas associated with epizootic hosts are heavily infected with bacilli, which block the foregut and are regurgitated into feeding wounds when fleas attempt to feed. Humans are infected when they intrude into epizootic habitats or when commensal rodents near housing are involved. Cats that kill and eat infected rodents or consume their carcasses have been known to transmit plague pneumonically, and plague may be acquired by direct contact with infected animal tissues.

Plague in the United States historically was limited to campestral cycles west of the 100th meridian. Recent findings, however, suggest a movement eastward, possibly into suburban and urban areas. For example, in March 1993, populations of fox squirrels near Abilene, Tex, were dying off as a result of plague. In May 1993, an infected fox squirrel and roof rat were collected in a residential area in Dallas County, Tex.[9]

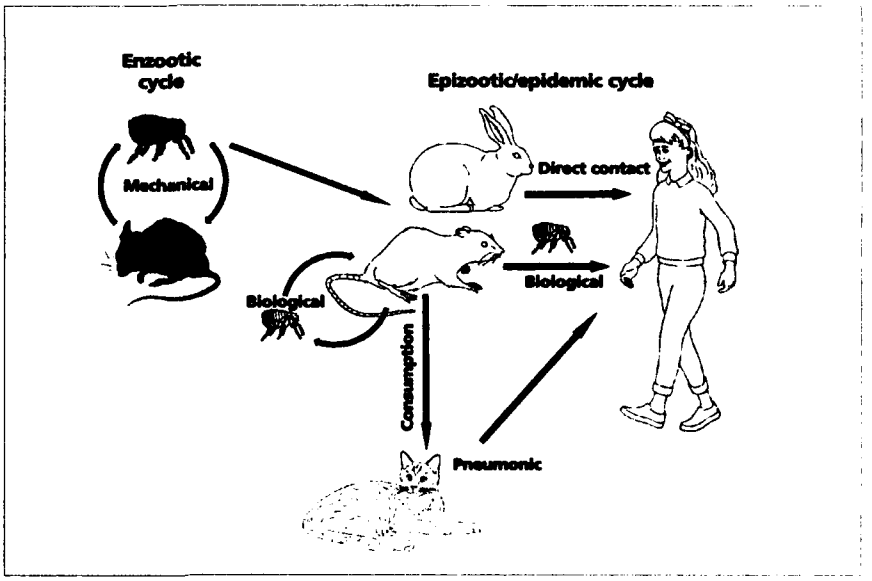

Fig 3.—Enzootic and epizootic transmission cycles of plague bacillus.

Tularemia

Francisella tularensis is another bacterium with a wide host range, several vectors, and a remarkable number of transmission mechanisms. Rabbits and some rodents, including muskrats, are important reservoir hosts in the United States. *Dermacentor* and other ixodid ("hard ticks") serve as biological vectors and, because transovarial transmission occurs in these species, reservoirs of the bacterium. West of the Mississippi River, transmission is biological, by tick bite, and mechanical, by biting flies (eg, deer flies). Transmission in the west occurs primarily in the summer and fall, the seasonal activity period of ticks and flies. In the eastern United States, arthropods still transmit tularemia, but most humans are infected when hunting rabbits and muskrats. Transmission is primarily through direct contact with infected tissues, and peak incidence occurs during winter hunting season. Humans also may be exposed by eating undercooked, infected meat, drinking contaminated water, inhaling bacteria in dust, or being bitten or licked by infected pets.

Leishmaniasis

Leishmania parasites are the most diverse group of protozoans pathogenic to humans. Leishmaniasis occurs in many ecological settings, from arid, rural areas, to urban environments and tropical forests. *Leishmania* causes a spectrum of human disease, from self-limited cutaneous lesions, to destruction of the nasal and oral mucosa, to widespread visceral involvement.

Human disease is uncommon in the United States (less than 30 locally acquired cases have been diagnosed) and is limited primarily to southern Texas. In this semi-arid region, the enzootic cycle involves transmission among wood rats (*Neotoma*)[10] by a sand fly, *Lutzomyia anthophora*, which inhabits nests of rodents (Fig 4).[11] Other vertebrate hosts and other sand fly species may be involved, but this has not been demonstrated.

Only adult, female flies bloodfeed, and the cyclopropagative cycle of the parasite is limited to the gut lumen of the fly. Damage to mouthpart valves allows parasites to leak out of the sand fly mouth and into the feeding wound.[12]

Humans are primarily infected when they live near or are active in the cactus-mesquite habitat of the wood rats. Because *L. anthophora* does not commonly feed on humans, a second sand fly species may act as a bridge from wood rats to humans. All isolates from humans, sand flies, rodents, and a single cat infection in Texas have

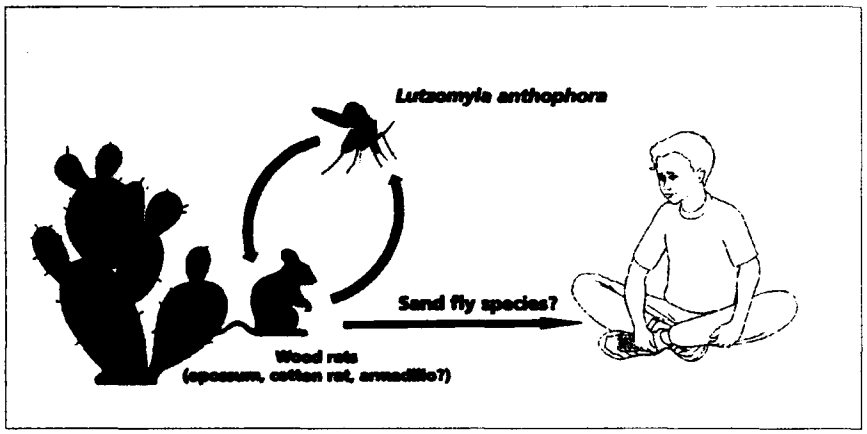

Fig 4.—Transmission cycle of *Leishmania mexicana* in Texas. Question mark indicates possible association.

been identified as *Leishmania mexicana*, a relatively benign species of parasite. Visceralizing infections in dogs have been reported from Texas, Oklahoma, and Ohio, but the source and identity of these parasites remain unclear.

Leishmaniasis occurs practically worldwide in a wide variety of vertebrate hosts. Human hosts, such as immigrants, travelers, or military personnel, or other hosts, such as dogs, entering the United States from endemic areas could potentially introduce exotic species of the parasite. Areas of the United States with anthropophilic (human-biting) sand flies such as *Lutzomyia diabolica* are at a particular risk for introductions.

Human Immunodeficiency Virus and Arthropods

Because human immunodeficiency virus (HIV) is a blood-borne pathogen, concerns have been raised about the possible transmission of HIV by blood-feeding arthropods. Laboratory studies and epidemiologic surveys indicate that this possibility is extremely remote. For biological transmission, the virus must avoid digestion in the gut of the insect, recognize receptors on and penetrate the gut, replicate in insect tissue, recognize and penetrate the insect salivary glands, and escape into the lumen of the salivary duct. In one study by Webb and colleagues, the virus persisted for 8 days in bedbugs.[13] Another study by Humphrey-Smith and colleagues showed the virus to persist for 10 days in ticks[14] artificially fed meals with high levels of virus ($>10^5$ tissue culture infective doses/mL [TCID/mL]), but there was no evidence of viral replication. Intra-abdominal inoculation of bedbugs and intrathoracic inoculation of mosquitoes was used to bypass any gut barriers, but again the virus failed to multiply.[13] Likewise, in vitro culture of HIV with a number of arthropod cell lines indicated that HIV was incapable of replicating in these systems. Thus, biological transmission of HIV seems extremely improbable.

Mechanical transmission would most likely occur if the arthropod were interrupted while feeding, and then quickly resumed feeding on a susceptible host. Transmission of HIV would be a function of the viremia in the infected host and the virus remaining on the mouthparts or regurgitated into the feeding wound. The bloodmeal residue on bedbug mouthparts was estimated to be 7×10^{-5} mL, but 50 bedbugs, interrupted while feeding on blood containing 1.3×10^5 TCID/mL HIV, failed to contaminate the uninfected blood on which they finished feeding or the mouse skin membrane through which they refed.[13]

Within minutes of being fed blood with 5×10^4 TCID of HIV, stable flies regurgitated 0.2 µL of fluid containing an estimated 10 TCID.[15] The minimum infective dose for humans contaminated in this manner is unknown, but under conditions such as those in some tropical countries where there are large populations of biting insects and a high prevalence of HIV infection, transfer might be theoretically possible, if highly unlikely. In these countries, however, other modes of transmission are overwhelmingly important, and, although of fatal importance to the extremely rare individual who might contract HIV through an arthropod bite, arthropods are of no significance to the ecology of this virus.

An epidemiologic survey of Belle Glade, a south Florida community believed to have a number of HIV infections in individuals with no risk factors, provided no evidence of HIV transmission by insects.[16] Interviews with surviving patients with the infections revealed that all but a few had engaged in the traditional risk behavior (eg, drug use and unprotected sex). A serosurvey for exposure to mosquito-borne viruses demonstrated no significant association between mosquito contact and HIV status. Nor were repellent use, time outdoors, or other factors associated with exposure to mosquitoes related to risk of HIV infection. A serosurvey for HIV antibodies detected no positive individuals between 2 and 10 years of age or 60 and older. No clusters of cases occurred in houses without other risk factors. There was thus no evidence of insect-borne HIV transmission.

Arthropods in a Clinical Laboratory Setting

Attending physicians or concerned patients may submit arthropods to the laboratory for identification and screening for pathologic agents. Most often, these are easily collected arthropods, such as lice, ticks, and fleas. Identification is the more important of these two requests. Specimens should be handled and preserved as detailed by Lago and Goddard[17] in the May issue of *Laboratory Medicine*. In some cases, referral to a medical entomologist or a specialist in a particular taxonomic group may be necessary for a definitive identification. A notation in the patient's

medical record is appropriate, particularly when the specimen is determined to be an important vector species. A specific identification and a thorough history, including travel and outdoor activities, will establish the potential for exposure and may prove invaluable in a differential diagnosis.

The screening of arthropods for etiologic agents, an important tool in investigations of the natural history of disease agents, is of limited value in a clinical setting. A negative test is no guarantee that an asymptomatic individual will stay healthy. The patient may have been bitten by other, infected arthropods. The parasitemia or viremia in the arthropod may have been below the detection level of the test, or the arthropod may have been infected with agents for which screening was not available or not conducted.

Conversely, detection of a positive arthropod does not necessarily indicate impending illness. The diagnostic test may cross-react and detect undescribed, nonpathogenic organisms. Although a pathogen may be present in the gut or body tissues of an arthropod, barriers to its transmission may render the species an inefficient or incompetent vector. The infected arthropod may be an efficient vector, but the pathogen may not have completed its developmental or propagative cycle, or there may be a delay between attachment and transmission. For example, transmission of *Borrelia burgdorferi* by *Ixodes scapularis* generally begins only after 48 hours of attachment.[18] Prophylactic treatment of healthy individuals exposed to infected arthropods is controversial and best determined on a case-by-case basis.

An infected arthropod found on a symptomatic patient may have acquired its infection from the patient rather than vice versa. In this situation, the positive specimen may serve to confirm other diagnostic tests. Because the arthropod may also have acquired its infection elsewhere, however, xenodiagnosis based on field-acquired arthropod infestations is by itself unreliable.

Conclusions

A variety of medically important pathogens are transmitted by an equally diverse array of arthropod vectors. The relationship between pathogen and arthropod may involve simple mechanical transport or the pathogen may undergo multiplication, or development, or both in the arthropod. A number of ABDs occur in the United States and illustrate the complex, dynamic nature of these transmission cycles. These diseases have the potential to become more important should conditions change. Human immunodeficiency virus is not likely to be transmitted by arthropods. When presented a possible vector in the laboratory, the most important task is identification. ❑

Acknowledgements

The author thanks Dr DT Dennis, Dr RS Nasci, Dr JG Rigau, Dr JR Zucker, and Ms J Dawson of the Centers for Disease Control and Prevention for providing the incidence data in the table. The manuscript for this article was reviewed by Dr J Goddard, Dr ML Womack, and Dr PC Melby.

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